A STUDY OF THE EFFECTS OF VARIOUS LEVELS OF CRANIALLY APPLIED ELECTRICITY ON PASSAGE OF GLUCOSE FROM CEREBRAL CIRCULATION INTO THE BRAIN

An abstract of a Thesis by
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The problem. Electricity, as a controlled energy, has been utilized in many capacities with varying degrees of success. Among these applications is Electroanesthesia (EA). Much is known about the gross effects induced on physiological systems while a subject is experiencing EA, but little is known about the effects on glucose transport into the brain.

Procedure. Thirty male rats were subdivided into 6 groups of 5 each, 1 serving as the control and the remaining 5 as experimental. The right common carotid artery was surgically exposed in all rats and a solution of ^{3}HOH, Glucose-U-^{14}C, and physiological saline was injected cephalically with decapitation 15 seconds later. The right rostral, ipsilateral portion of the brain was then extracted and prepared for scintillation counting. The experimental groups received 1, 4, 8, 12, and 16 milliwatts respectively for 3 minutes and 45 seconds prior to injection. Nine separate counts were run on each sample of brain tissue.

Findings. Examination of the ^{3}H and ^{14}C disintegrations per minute showed no difference between the control and experimental groups in uptake of the Glucose-U-^{14}C as compared to the freely diffusing ^{3}HOH indicator.

Conclusions. Statistical analysis of the resulting data indicates no significant change of glucose passage across the blood-brain barrier into the brain tissue between control and experimental groups.

Recommendations. The parameters of this study did not include the effects produced by electricity at levels other than the 5 investigated. Future studies should encompass a wider scope of electricity levels, regulation of the injection speed, and use of a chemical anesthetic which does not depress metabolism.

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A Thesis

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In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

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by

Craig Robert Evans

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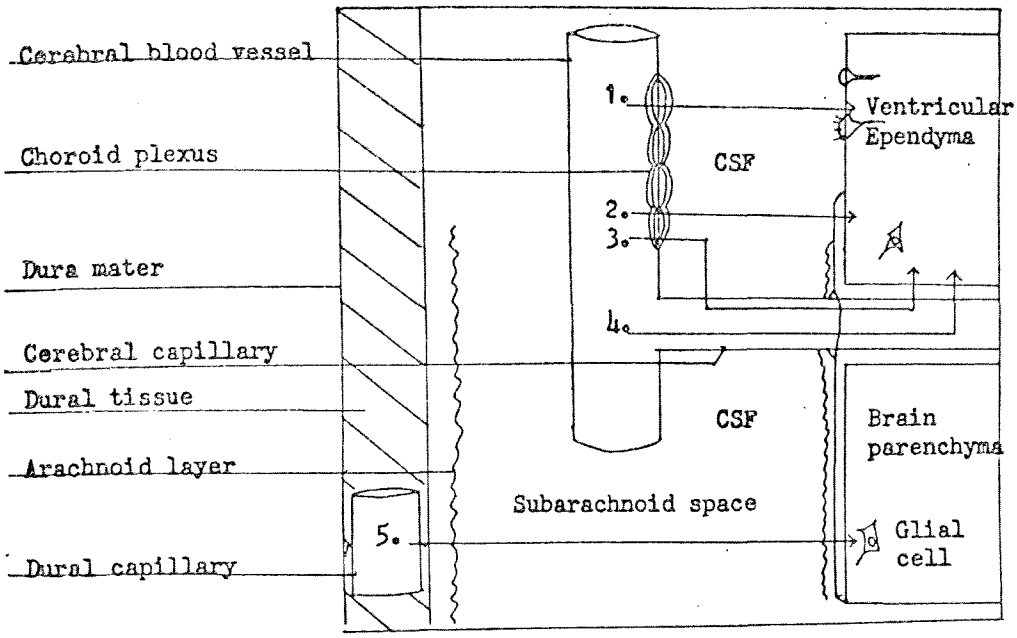
INTRODUCTION

Blood-Brain Barrier. For many years the concept of a blood-brain barrier has been explored and described in search of a thorough understanding of its function. Ehrlich (1887, cited by Davson, 1972) in his studies of vital staining, noted that when an animal was injected intravenously with a variety of dyes all tissues were stained while the brain, but not the dura, was spared. At the turn of the century a second early researcher, Lewandowsky (1900, cited by Davson, 1972), showed that the Prussian blue reagents failed to pass from the circulation into the brain and spinal cord. From this information he formulated clearly the concept of a barrier that presents an impediment to the passage of materials from cerebral circulation into the brain. In 1909, Goldman (1909 and 1913, cited by Davson, 1972) injected trypan blue into the blood and noticed that the brain was not stained and the dye did not enter the cerebrospinal fluid. In a second experiment four years later he injected the dye directly into the cerebrospinal fluid. This time the entire brain took up the dye. It appeared that the ependymal linings of the ventricles and the pial-glial linings to the brain parenchyma were ineffective barriers to the passage of trypan blue from cerebrospinal fluid into the brain.

The selective permeability of the blood-brain barrier

has been closely analyzed with respect to which substances are allowed to pass and the manner by which they pass. Recent investigations into the blood-brain barrier (Davson, 1972; Holman, 1972) have detected the selective transfer of essential substances across the barrier into the brain by mechanisms other than simple diffusion. The transfer of glucose into the brain is necessary for any animal's existence. Crone (1965) postulated that glucose crossed the barrier not only by diffusion but also by a carrier mediated transfer system. Exactly what microstructures are responsible for these systems of transfer are unknown. The findings of Eidelberg et al. (1967) and Gilbert (1965) also suggest the possibility that the transport of sugars into the brain may be active and require metabolic energy. Other work done by Rosenberg and Wilbrandt (1956), Bowyer (1957), Morgan et al. (1964), Regen and Morgan (1964), Crawford (1967), and Buschiazzo et al. (1970) leads to the conclusion that the facilitating mechanism by which glucose crosses the blood-brain barrier involves a mobile carrier similar to that which transports glucose into erythrocytes, muscle, and adipose tissue. Figure 1 illustrates several postulated routes by which materials from the blood enter the brain.

Electroanesthesia. Since the introduction of electricity as a controlled energy, experiments have been performed to determine the gross effects on physiological systems



1. Blood → Choroid plexus → CSF → Ependyma of ventricles → brain

2. Blood → Choroid plexus → CSF → Pial-glial membranes → brain

3. Blood → Choroid plexus → CSF → Cerebral capillary → Extracellular space → Extracellular fluid → brain

4. Blood → Extracellular space → Extracellular fluid → brain

5. Dural capillaries → Dural tissue → CSF → Pial-glial membranes → brain

Figure 1. Postulated blood to brain molecular transport routes.

caused by passage of electricity. Cranially applied electricity may produce any of 3 phenomenon. These phenomenon are described as Electrosleep--a sleep like state, Electroanalgesia--the absence of pain, and Electroanesthesia--the absence of all sensation. Of the 3, Electroanesthesia (EA) is thought to be the most promising for applications to mammals. The concept of using electricity to produce anesthesia is not by any means new. Benjamin Franklin performed experiments in this direction in the 18th century. The first published research employed the use of direct current to attain electroanesthesia. Mach (1875, cited by Herin, 1968) produced a level of unconsciousness in fish by passing a direct current through an aquarium. In larger animals, experiments were usually hampered by the various degrees of muscle rigidity, convulsive states, and burning produced by the direct current. The earliest record of an attempt to produce sleep in a human with electricity was by LeDuc and Rouxeau (1902, cited by Herin, 1968). Soon after, Rouxeau, LeDuc and Robinovitch began the initial investigations into the physiological effects produced by electricity. In 1910, EA was used successfully in surgery during amputation of a patient's toes. In 1934, the introduction of alternating current for the induction of anesthesia brought new advantages to the field. Among the advantages were a decreased tendency for tissue destruction when needle and disc electrodes were used, the minimization of

cellular polarity due to the alternating cycle of the current, a constant ratio of average current to peak current, and the opportunity to monitor EEG patterns (Collins, 1966; Smith, 1967, 1971).

No definite pattern of administration has been determined. Electrodes are generally attached bitemporally on mammals, but there are several other attachment areas. EA is usually induced after a small amount of chemical tranquilizer has been administered to quiet the animal.

EA today has been used successfully in many countries for surgeries ranging from minor suturing to ovariohysterectomies. Although many physiological effects produced by EA are known, much more investigation is needed. In a recent review article by Robert Smith on Electroanesthesia (1971), a list of known physiological effects produced on various mammals during EA was presented. Here is a summary of that list.

1. Cerebral blood flow is unchanged.
2. Cerebral O_2 consumption is not increased.
3. No change in brain gross, micro, or ultrastructure following EA. (Conflicting evidence was found with fact #3 by Sances et al. (1966).)
4. No change in ability of dogs to perform learned tricks or to learn new ones.
5. No obstruction of the airway during EA.
6. Blood gasses remain normal throughout.
7. Blood pressure does not fall during EA, and there is no postural hypotension. Blood elicites normal compensatory mechanisms up to shock levels, precisely as though the animal were awake.
8. Body temperature stays under control.

Fabian et al. (1964), Geddes et al. (1964), and

Stratton (1974) cited additional physiological effects including significant increases in the catacholamine, free and conjugate corticoid and blood sugar levels suggesting a sympathoadrenal response to the electricity. An increase in gastrointestinal activity, salivation, micturition, defecation and in some cases convulsions were other noted physiological changes. Of more interest is a significant increase of glucose in cerebral spinal fluid.

There are two major theories that offer explanations on how centrally acting EA works. The first, as defined by Price and Dornette (1963), describes a "jamming" of the neuronal network by the alternating current. It is postulated that the passage of the current through the thalamus blocks the classical sensory pathways that reach the higher cortical centers via synapses in the thalamus, between the second and third neurons of the 3-neuron sensory pathways. Repolarization of the various nerve pathways is then prevented, as is further conduction of impulses over these pathways. A second theory, Anokin (1969) and Magnes et al. (1973), involves an inhibition of the reticular activating system and a simultaneous blocking, by polarization, of the classical ascending sensory pathways. The mechanism or mechanisms by which electricity produces analgesia, sleep and anesthesia is still a mystery. This project will not attempt to answer this question.

Since glycogen stores in the brain are not extensive,

a constant supply of this principal nutrient is necessary. If EA were to inhibit glucose passage across the barrier into the brain, deterioration of brain parenchyma in the form of glial cells and neurons would be imminent. Likewise, if electricity were to increase the passage of glucose into the brain, possible injury in the form of cerebral edema (Bakay and Lee, 1965; Klatzo, 1967), with subsequent compression of blood vessels and brain cells might eventually lead to deterioration of the brain and the animal.

If anesthesia in the form of bitemporally applied electricity is to be used routinely on living systems in the future, it is essential to know the answers to several significant questions. This project is designed to answer one of them. Does anesthesia in the form of bitemporally applied electricity influence glucose transport from cerebral circulation into the brain?

MATERIALS AND METHODS

Thirty male rats, approximately 250-300 grams, were first randomly subdivided into six groups of five each. Group 1 served as the control group and groups 2 through 6 received 1, 4, 8, 12, and 16 milliwatts respectively. The five variable powers investigated in the experiment were accepted physiological levels used for induction and maintenance of EA in rats.

Group 1 - Controls. Each rat was brought to a surgical stage of anesthesia with an intraperitoneal injection of Nembutal (40 mg/kg). The rat was then fastened firmly, with its ventral side exposed, to a specially constructed restraint board measuring approximately 10 inches by 14 inches. It was secured by means of strings to hook devices placed 2 on each side of the board at the level of the rat's appendicular girdles as illustrated in figure 2. The right common carotid artery was surgically exposed and cleared of all surrounding materials until approximately 3/4 to 1 inch was showing. A 0.2 ml mixture containing 0.5 µc Glucose-U-^{14}C (labeled glucose), 2.0 µc ^{3}HOH (labeled water) (New England Nuclear Corp., Boston, Mass.), in mammalian Ringer's solution, buffered to pH 7.4 at approximately 22 C was then injected cephalically using a 27½ gauge, ½ inch needle, as rapidly as possible. The needle was left in the artery to avoid excessive bleeding. Care was taken to avoid obstruction of the artery so that blood flow would continue throughout the needle's placement in the artery.

Exactly 15 seconds after the injection, enough time for 1 passage of blood through the brain but not enough for a second (Oldendorf, 1970), the animal was decapitated and the right rostral (ipsilateral) portion of its brain removed. Upon removal, the brain was cleared of all surrounding matter and blood vessels since these could be a cause of scintillation quenching. The brain was placed into

a scintillation vial containing 0.1 ml Soluene (tissue solubilizer, Packard Instruments Co. Inc.). When the brain had been completely dissolved, 10 ml Aquasol (scintillation fluor, Beckman Instruments Inc.) was added and the sample was placed in the scintillation machine (Beckman model LS-100 C., Beckman Instruments Inc.). In order to minimize chemiluminescence, the sample was allowed to stand in the machine for at least eight hours prior to counting. The entire process was then repeated with the remaining 4 rats in this group.

Groups 2 through 6 - Experimental. The process was repeated as in group 1 with the addition of cranially applied electricity. Two alligator clip electrodes were attached bitemporally to the depilitated skin with electrode paste to insure a positive contact. Correct placement of bitemporal electrodes is illustrated in figure 3. For the rats in group 2 a 1 mW level of power from the Stoelting Electroanesthesia device (Stoelting Manufacturing Co.), was then passed through the brain for 3 minutes and 45 seconds prior to the bolus injection. The injection was administered exactly 15 seconds before the end of the 4 minute EA induction period. Upon termination of the induction the animal was decapitated and the brain prepared for scintillation counting as in group 1.

Groups 3, 4, 5, and 6 were treated as in group 2 using 4 mW, 8 mW, 12 mW, and 16 mW respectively. As

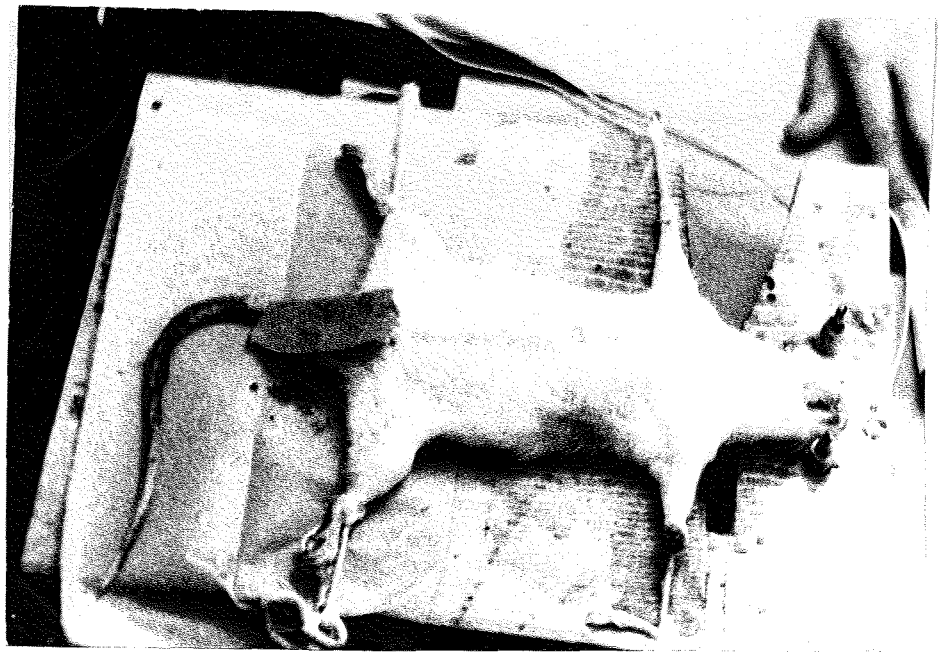


Figure 2. Proper mounting of a rat on a restraint board.

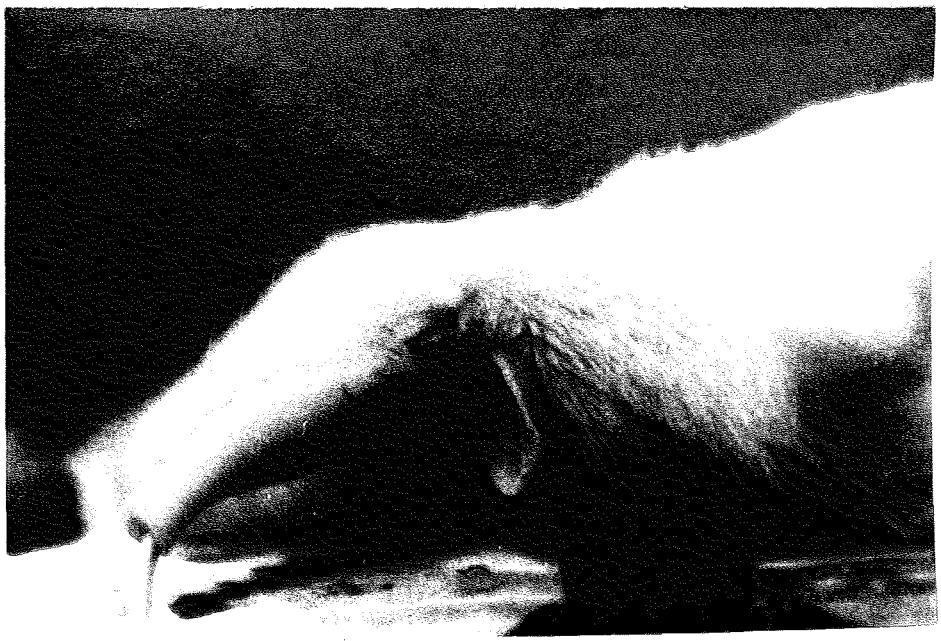


Figure 3. Correct placement of bitemporal electrodes.

indicated, the Stoelting Electroanesthesia device is used to apply power to rats in groups 2 through 6. The device contains meters to indicate the RMS voltage applied and RMS current that passes. Power was calculated by multiplying the voltage by the amount of current passed. $I \times E = P$. The appropriate amounts of power were obtained by careful manipulation of the voltage and the current.

The samples were then analyzed to show the amount of labeled glucose and labeled water present. The amount of each isotope present is directly related to the number of counts per minute (CPM) when the counting efficiency is considered. Both isotopes are beta emitters. The scintillation counter is capable of distinguishing between them.

The ^{3}HOH injected into the common carotid artery which flows into the internal carotid artery is distributed to the brain from the blood in the course of 1 passage through the cerebral capillaries (Oldendorf, 1970, 1971; Yudilevich and DeRose, 1971). An unknown fraction of the labeled glucose also passes into the brain. That portion of the labeled glucose not taken up by the brain is carried out of the cerebral circulation before decapitation.

Counts per minute (CPM) were converted to disintegrations per minute (DPM). The number of DPM is directly related to the amount of isotope present. This conversion involves quench correction techniques using the external standard-channels ratio method and quench correction curves

in order to calculate the counting efficiencies. Appendix Table 1 is a list of derived values used as counting efficiencies.

In order to measure the transfer of labeled glucose, it was necessary to calculate an index of uptake of the isotopes.

The labeled glucose to labeled water DPM ratio in the brain sample is divided by the labeled glucose to labeled water DPM ratio in the injection mixture and the result multiplied by 100 in order to provide the amount of labeled glucose taken up by the brain as a percentage of the labeled water extracted. This ratio, known as the Brain Uptake Index (BUI) (Oldendorf, 1971) is then used to calculate differences, if any, between the experimental rats and the control rats.

$$BUI = \frac{\text{tissue Glucose-U-}^{14}\text{C DPM/tissue }^{3}\text{HOH DPM}}{\text{injected Glucose-U-}^{14}\text{C DPM/injected }^{3}\text{HOH DPM}} \times 100.$$

RESULTS

Nine scintillation counts were run per sample in order to attain consistent CPM. "t" tests were performed using the BUI's to determine if significant differences from the control group of rats were present. The resulting t-value of 1.39 showed no significant difference between the rats in group 1, the controls, as compared to the rats in group 2

Table 1. Statistical analysis of resulting Brain Uptake Indexes (BUI).

	Control	Experimental				
	0 mW	1 mW	4 mW	8 mW	12 mW	16 mW
	Group 1	Group 2	Group 3	Group 4	Group 5	Group 6
BUI	56.66%	114.18%	6.09%	61.89%	40.10%	39.07%
	37.62%	172.61%	143.00%	4.20%	96.65%	67.99%
	115.28%	208.67%	103.36%	65.12%	731.39%	42.14%
		89.78%			72.28%	
		38.35%				
N=	3	5	3	3	4	3
$\bar{X}$=	69.85%	130.72%	84.15%	43.73%	235.11%	49.73%

$s^2_{(G_1, G_2)}$ = 3,595.87

t-value = 1.39

$s^2_{(G_1, G_3)}$ = 3,304.33

t-value = 0.31

$s^2_{(G_1, G_4)}$ = 1,410.39

t-value = 0.84

$s^2_{(G_1, G_5)}$ = 66,659.84

t-value = 0.84

$s^2_{(G_1, G_6)}$ = 949.08

t-value = 0.79

All t-values obtained showed no significant difference.

receiving 1 mW. The experimental rats in group 3 receiving 4 mW when compared to the rats in group 1 showed no significant t-value difference, 0.31, from the rats in group 1. Groups 4, 5, and 6 receiving 8 mW, 12 mW, and 16 mW respectively revealed 0.84, 0.84, and 0.79 for t-values confirming no significant change from the control rats.

Nine vials were eliminated from t test analysis due to major inconsistencies in scintillation analysis.

DISCUSSION

EA in the form of cranially applied electricity causes an increase in blood sugar (Yudilevich and DeRose, 1971) and an increase in glucose concentrations in CSF (Stratton, 1974). It has been postulated that all 5 routes of entry into the brain involve passage across the CSF. A question might be, would it not seem possible that an increase in CSF glucose would increase the concentration gradient between the CSF and brain parenchymal tissue resulting in a higher diffusion rate of glucose into the brain tissues? Electric power at the five investigated levels showed no change in passage of glucose from the cerebral circulation into the brain.

In discussing hypotheses for the effects of electricity on glucose passage into the brain, it is important to recall the recent evidence indicating a carrier transport mechanism for glucose across the blood-brain barrier. It

is conceivable that the rate of this transfer is dependent on the metabolic needs of the brain cells. Since the brain cannot concentrate its chief nutrient, it must supplement its requirements as needed. If the rate of glucose transfer is dependent on the metabolic needs of the brain cells it could be concluded that the electricity did not cause a major change in the brain's metabolic rate since glucose transfer did not increase.

There is also the question of when, if ever does the effect of electricity occur? It is possible that the amount of time allowed for induction was enough for the brain to homeostatically recover from the initial trauma induced by the electricity. Along with this thought it is also conceivable that the induction time was not long enough for any slower acting response to the electricity that might affect glucose passage into the brain.

The amount of labeled glucose passing into the brain appears to be proportional to the amount of labeled water diffusing into the brain. Glucose exhibits carrier saturation characteristics in high concentrations (Davson, 1972). If transport of glucose were confined to this carrier mechanism alone, a limit to the amount of glucose that could be transported per unit time would be reached. The linear regression line of figure 4 indicates that the carrier saturation point of glucose has not been attained. Electricity, at the five increasing intervals of power showed no

increase or decrease of the indicators passage into the brain. This does not rule out the possibility of affecting the glucose concentration in the brain by some means of stimulation other than the five investigated levels of power.

The wide variation of BUI percentages within each group might be attributed to the speed of the bolus injection. The slower the rate of injection, the higher the dilution of the bolus by the blood. Dilution of the injection creates competition among the injected labeled glucose and the unlabeled glucose found in the blood. This competition results in a decreased uptake of the labeled glucose as compared to an increased uptake of the more heavily concentrated unlabeled glucose normally present. Since the labeled glucose is the only glucose isotope counted by the scintillator, all other glucose passing the barrier into the brain would not be recorded. A rapid injection would result in a high concentration of labeled glucose in the brain parenchyma due to the bolus passage through the cerebral circulation in an undiluted state. The compact condition of the bolus would minimize glucose carrier site competition between the labeled and unlabeled glucose. Labeled water DPM in the scintillated brain tissue are proportional to blood flow (Oldendorf, 1971). Since labeled water passage across the blood-brain barrier is approaching 100% (Oldendorf, 1971) it is safe to assume all labeled water passing the

barrier sites enters the brain. It is also reasonable to assume that labeled water transfer is dilution independent.

The parameters of this study did not include the effects produced by electric power at levels other than the five investigated. It therefore cannot be concluded that other levels of power when applied cranially to a living mammalian system will have no effect on glucose passage into the brain of that system.

SUMMARY AND CONCLUSIONS

Analysis of the resulting data indicates no significant change of glucose passage into the brain across the blood-brain barrier while the rat is experiencing five levels of cranially applied electric power.

Nembutal is a known metabolism depressor. It is conceivable that the metabolic rate of the brain under the influence of nembutal was already so low that a further decrease is difficult to show. If the rate of glucose transfer is dependent on the metabolic needs of the brain cells, it can be concluded that the electricity did not involve an increase in the brain's metabolic rate.

The uncontrolled variable of injection speed might have lead to a masking of any possible effects produced by the electricity. If the injection time had been rapid and consistent, any change produced by electricity on glucose

passage into the brain would not have been masked. In future studies of this nature it is essential to regulate this variable.

APPENDIX

APPENDIX

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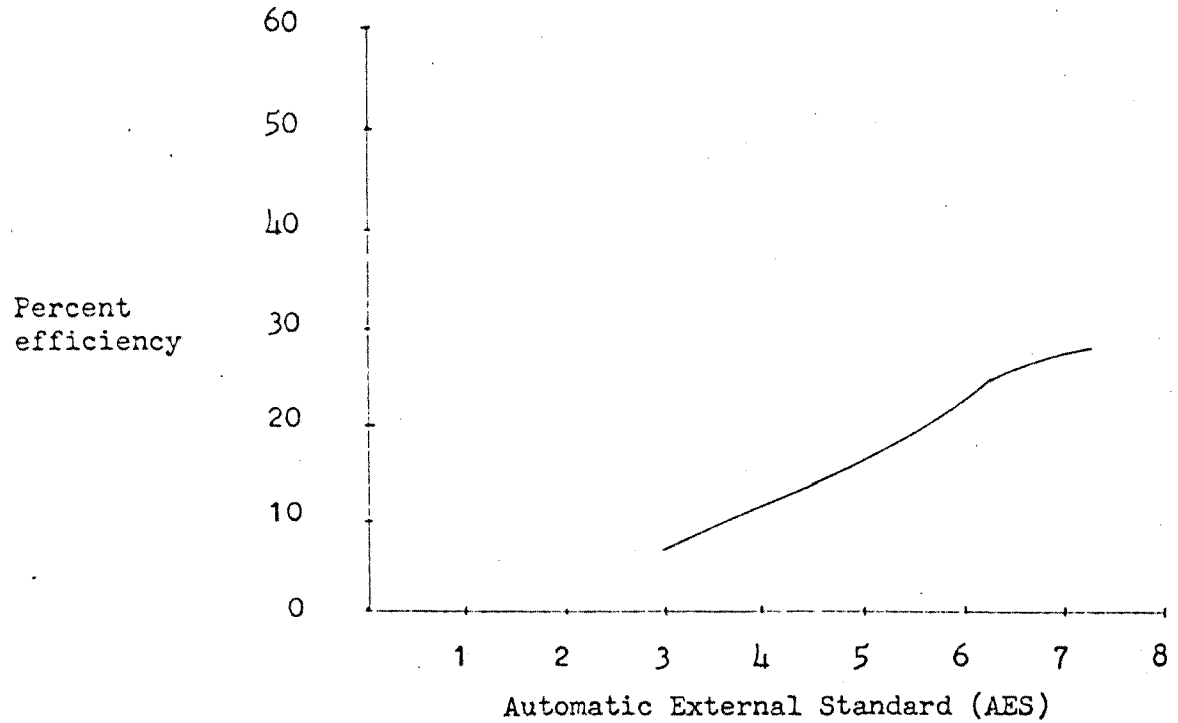

Figure 1. Quench Correction Curve showing ^{3}H DPM in the ^{3}H window. This graph is used to determine the percentage of ^{3}H DPM in the sample tissue being counted in the ^{3}H window. The Automatic External Standard is a measurement of the degree of sample quenching.

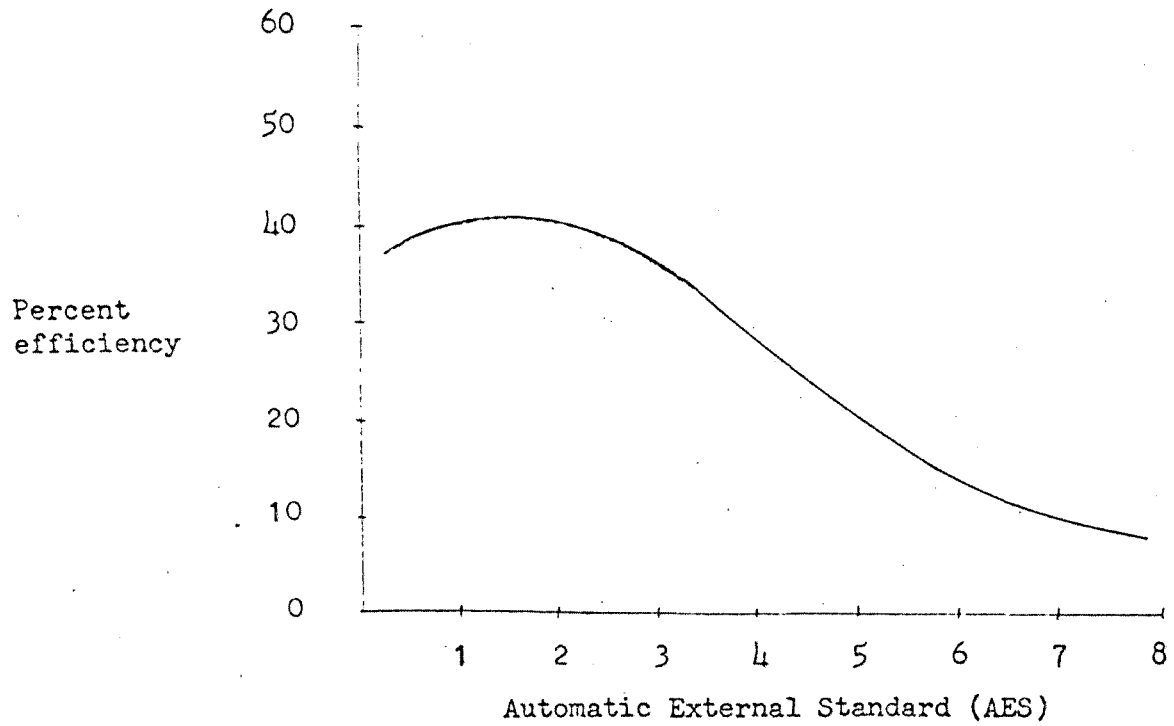

Figure 2. Quench Correction Curve showing ^{14}C DPM in the ^{3}H window. This graph is used to determine the percentage of ^{14}C DPM in the sample tissue being counted in the ^{3}H window. The Automatic External Standard is a measurement of the degree of sample quenching.

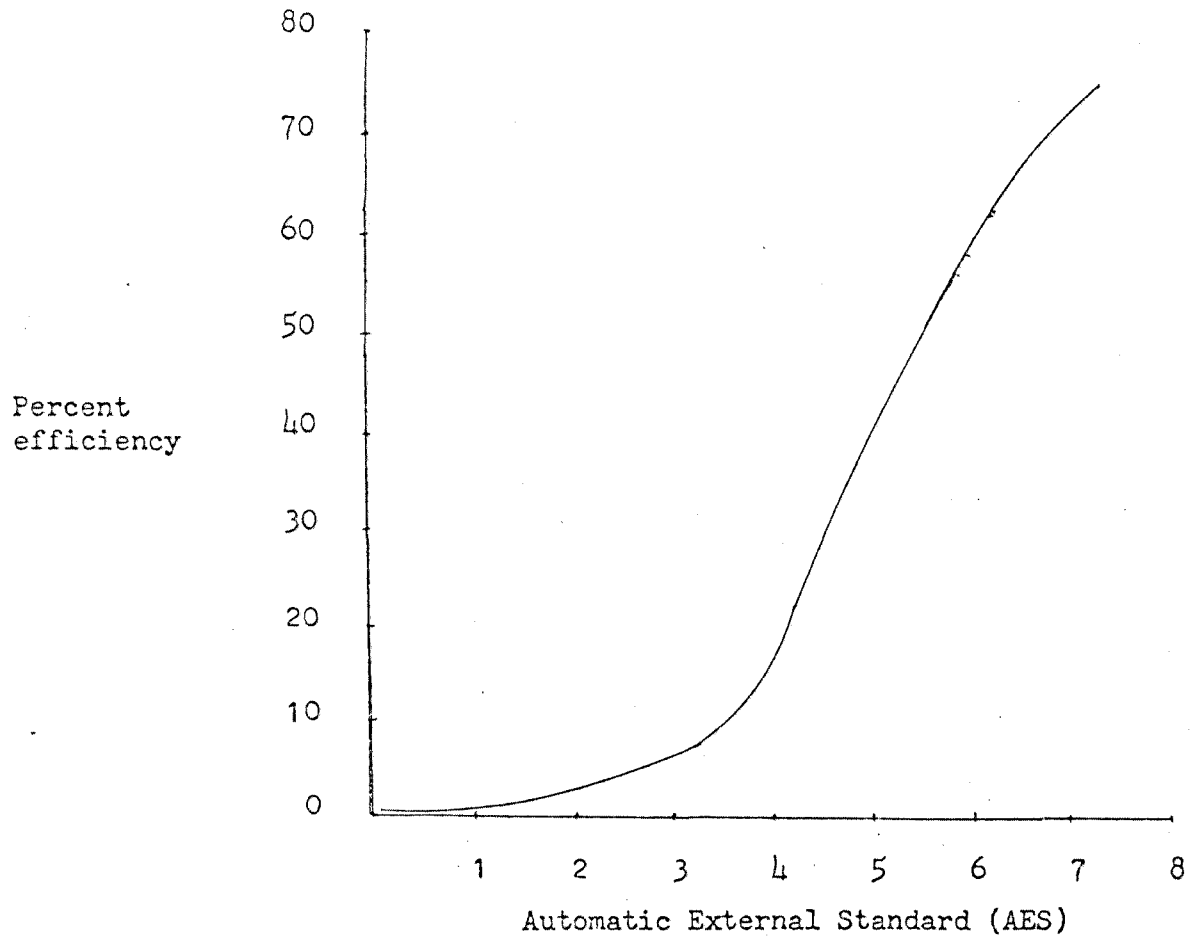

Figure 3. Quench Correction Curve showing ^{14}C DPM in the ^{14}C window. This graph is used to determine the percentage of ^{14}C DPM in the sample tissue being counted in the ^{14}C window. The Automatic External Standard is a measurement of the degree of sample quenching.

Table 1. Quench Correction Efficiency Values

AES	$^{3}H/^{3}H$	$^{14}C/^{3}H$	$^{14}C/^{14}C$	AES	$^{3}H/^{3}H$	$^{14}C/^{3}H$	$^{14}C/^{14}C$	AES	$^{3}H/^{3}H$	$^{14}C/^{3}H$	$^{14}C/^{14}C$
4.50	.137	.233	.290	4.90	.162	.203	.369	5.30	.189	.176	.440
4.51	.137	.233	.291	4.91	.163	.202	.371	5.31	.190	.175	.442
4.52	.138	.232	.292	4.92	.164	.201	.373	5.32	.191	.175	.443
4.53	.138	.232	.292	4.93	.164	.201	.375	5.33	.191	.174	.445
4.54	.140	.231	.298	4.94	.165	.200	.377	5.34	.192	.174	.447
4.55	.141	.230	.300	4.95	.165	.199	.379	5.35	.193	.173	.449
4.56	.142	.229	.302	4.96	.166	.198	.381	5.36	.194	.173	.450
4.57	.143	.228	.304	4.97	.167	.197	.383	5.37	.194	.172	.452
4.58	.143	.227	.306	4.98	.168	.197	.385	5.38	.195	.172	.453
4.59	.144	.227	.308	4.99	.169	.196	.387	5.39	.196	.171	.455
4.60	.144	.226	.310	5.00	.169	.195	.388	5.40	.197	.170	.456
4.61	.145	.225	.313	5.01	.170	.195	.390	5.41	.197	.170	.457
4.62	.145	.224	.315	5.02	.170	.194	.392	5.42	.198	.169	.459
4.63	.146	.223	.317	5.03	.171	.194	.394	5.43	.199	.169	.461
4.64	.146	.222	.319	5.04	.171	.193	.396	5.44	.200	.168	.462
4.65	.147	.221	.321	5.05	.172	.193	.398	5.45	.200	.168	.463
4.66	.147	.220	.323	5.06	.172	.192	.399	5.46	.201	.167	.456
4.67	.148	.220	.325	5.07	.173	.191	.400	5.47	.202	.167	.467
4.68	.148	.219	.327	5.08	.174	.190	.401	5.48	.203	.166	.468
4.69	.149	.218	.329	5.09	.174	.190	.402	5.49	.204	.166	.470
4.70	.149	.218	.332	5.10	.175	.189	.494	5.50	.204	.165	.472
4.71	.150	.217	.334	5.11	.176	.188	.407	5.51	.205	.164	.474
4.72	.151	.216	.336	5.12	.177	.187	.409	5.52	.206	.163	.475
4.73	.151	.215	.338	5.13	.177	.187	.411	5.53	.207	.163	.477
4.74	.152	.215	.340	5.14	.178	.186	.413	5.54	.207	.162	.478
4.75	.152	.214	.341	5.15	.179	.186	.415	5.55	.208	.161	.479
4.76	.153	.213	.343	5.16	.180	.185	.416	5.56	.208	.161	.480
4.77	.153	.213	.345	5.17	.180	.185	.418	5.57	.209	.160	.482
4.78	.154	.212	.347	5.18	.181	.184	.420	5.58	.210	.160	.484
4.79	.154	.211	.349	5.19	.182	.183	.422	5.59	.210	.159	.486
4.80	.155	.210	.350	5.20	.183	.182	.423	5.60	.211	.158	.488
4.81	.156	.209	.352	5.21	.183	.181	.425	5.61	.211	.158	.490
4.82	.157	.209	.354	5.22	.184	.181	.427	5.62	.212	.157	.492
4.83	.157	.208	.356	5.23	.184	.180	.429	5.63	.212	.156	.493
4.84	.158	.207	.358	5.24	.185	.180	.430	5.64	.213	.156	.494
4.85	.158	.206	.360	5.25	.186	.179	.432	5.65	.214	.155	.496
4.86	.159	.206	.362	5.26	.187	.179	.434	5.66	.215	.155	.497
4.87	.160	.205	.364	5.27	.188	.178	.435	5.67	.217	.154	.498
4.88	.161.	.204	.366	5.28	.188	.177	.436	5.68	.218	.154	.500
4.89	.161	.204	.367	5.29	.189	.176	.438	5.69	.220	.153	.502

The efficiency values were derived from the Quench Correction Curves found in the appendix. Values expressed are a ratio of counts per minute of the isotope present in a channel to the known disintegrations of the isotope. The Automatic External Standard (AES) is a measurement of the degree of sample quenching. Efficiency percentages are calculated by multiplying this ratio by 100.

Table 2. Individual sample information and data.

Group 1 - Controls		AES	CPM ^3H	CPM ^{14}C	Efficiencies $^{14}C/^{14}C$	Efficiencies $^{14}C/^3H$	Efficiencies $^3H/^3H$	DPM ^3H	DPM ^{14}C	Injection ratio $^{14}C/^3H$	BUI	Mean BUI
		5.56	39,126.92	9,060.00	.480	.161	.208	173,500.19	18,875.00		55.22%	
		5.42	39,319.23	9,513.20	.459	.169	.198	180,891.66	20,725.93		58.16%	
Rat weight	.344 gr	5.48	39,430.76	8,619.23	.468	.166	.203	179,179.85	18,417.16		52.18%	
Nembutal injected	.196 ml	5.37	39,669.23	8,572.32	.452	.172	.194	187,665.97	18.965.31		51.30%	
Brain extracted	.280 gr	5.48	38,273.07	9,318.51	.468	.156	.203	172,255.12	19,911.35	0.197	58.68%	56.66%
Vial number	3	5.43	39,019.23	9,959.04	.461	.169	.199	177,730.15	21,603.12		61.70%	
		5.47	39,466.15	9,171.42	.467	.167	.202	179,041.73	19,639.01		55.68%	
		5.45	38,953.84	9,085.98	.463	.168	.200	178,284.90	19,624.15		55.87%	
		5.41	39,423.07	9,984.25	.457	.170	.197	181,264.06	21,847.37		61.18%	
						.176						
		5.29	4,933.96	827.41	.438	.171	.189	24,346.51	1,889.06		39.39%	
		5.22	4,824.52	710.87	.427	.180	.184	24,582.55	1,664.80		34.38%	
Rat weight	.328 gr	5.24	4,717.84	827.49	.430	.181	.185	23,629.46	1,924.40		41.34%	
Nembutal injected	.187 ml	5.21	4,821.69	710.29	.425	.187	.183	24,695.03	1,671.27		34.35%	
Brain extracted	.310 gr	4.98	4,592.34	714.13	.385	.190	.168	25,160.30	1,854.88	0.197	37.42%	37.62%
Vial number	6	5.24	4,593.30	719.42	.430	.180	.185	23,200.81	1,673.07		36.61%	
		5.23	4,597.12	710.72	.429	.180	.184	23,363.70	1,656.69		35.99%	
		5.23	4,809.61	715.89	.429	.189	.184	24,506.74	1,668.74		34.57%	
		5.25	4,372.38	821.26	.432	.177	.186	21,677.90	1,901.06		44.52%	
						.186						
		5.15	15,375.00	6,532.91	.415	.185	.179	69,536.31	15,741.95		114.92%	
		5.16	15,072.30	6,874.37	.416	.185	.180	66,751.06	16,524.93		125.67%	
Rat weight	.323 gr	5.16	15,942.18	6,432.91	.416	.185	.180	72,674.39	15,463.73		108.01%	
Nembutal injected	.200 ml	5.17	15,284.61	6,993.20	.418	.180	.180	67,719.61	16,730.14		125.41%	
Brain extracted	.250 gr	5.23	15,286.15	6,998.14	.429	.182	.184	67,118.86	16,312.68	0.197	123.37%	115.28%
Vial number	31	5.20	15,659.09	6,091.19	.423	.189	.183	71,247.49	14,399.98		102.59%	
		5.26	15,835.38	6,778.39	.434	.171	.187	69,730.91	15,618.41		113.70%	
		5.22	15,977.27	6,447.85	.427	.181	.184	71,978.86	15,100.35		106.49%	
		5.22	15,763.63	6,889.50	.427	.188	.184	69,800.33	16,134.66		117.34%	

This table contains the general presurgical and postsurgical information of the sample, the resulting scintillation counts with an Automatic External Standard (AES), the derived Quench Correction Efficiencies and the calculated disintegrations per minute (DPM). The Brain Uptake Index (BUI) is determined by dividing the ratio of $^{14}C/^3H$ DPM found in the brain tissue by the $^{14}C/^3H$ DPM ratio in the injection mixture. The mean BUI is the sum of all BUI's divided by the nine separate counts.

Table 3. Individual sample information and data.

Group 2 - 1 milliwatt		AES	CPM ^3H	CPM ^{14}C	Efficiencies $^{14}C/^{14}C$	Efficiencies $^{14}C/^3H$	Efficiencies $^3H/^3H$	DPM ^3H	DPM ^{14}C	Injection ratio $^{14}C/^3H$	BUI	Mean BUI
		5.41	1,450.05	636.15	.457	.170	.197	6,159.44	1,392.01		114.72%	
		5.38	1,348.74	527.66	.453	.172	.195	5,889.18	1,164.81		100.40%	
Rat weight	.335 gr	5.47	1,452.05	742.86	.467	.167	.202	5,873.27	1,590.71		137.48%	
Nembutal injected	.191 ml	5.50	1,235.30	745.83	.472	.165	.204	4,777.35	1,580.15		167.90%	
Brain extracted	.220 gr	5.50	1,561.18	741.91	.472	.165	.204	6,381.52	1,571.84	0.197	125.03%	144.18%
Vial number	10	5.47	1,343.06	741.69	.467	.167	.202	5,335.79	1,588.20		151.09%	
		5.50	1,129.96	741.50	.472	.165	.204	4,268.38	1,570.97		186.83%	
		5.51	1,347.95	636.91	.474	.164	.205	5,500.39	1,343.69		124.01%	
		5.53	1,121.55	747.67	.477	.163	.207	4,183.86	1,567.44		190.17%	
		5.09	563.58	120.65	.402	.190	.174	2,911.26	300.12		52.33%	
		5.10	670.03	120.53	.404	.190	.174	3,506.51	298.34		43.19%	
Rat weight	.303 gr	5.08	891.15	120.97	.401	.190	.174	4,792.13	301.67		31.95%	
Nembutal injected	.173 ml	5.06	891.45	121.02	.399	.192	.172	4,844.24	303.31		31.78%	
Brain extracted	.340 gr	5.08	781.59	121.38	.401	.190	.174	4,161.38	302.69	0.197	36.92%	38.35%
Vial number	14	5.11	677.68	121.19	.407	.188	.176	3,532.39	297.76		42.79%	
		5.11	674.99	120.94	.407	.188	.176	3,517.78	297.15		42.88%	
		5.08	897.02	121.36	.401	.190	.174	4,824.83	302.64		31.84%	
		5.15	895.32	120.75	.415	.186	.179	4,699.44	290.96		31.43%	
		5.51	546.54	343.23	.474	.164	.205	2,086.78	724.11		176.14%	
		5.47	545.95	343.12	.467	.167	.202	2,095.30	734.73		178.00%	
Rat weight	.335 gr	5.48	549.12	343.51	.468	.166	.203	2,104.83	734.00		177.02%	
Nembutal injected	.191 ml	5.51	548.59	343.23	.474	.164	.205	2,096.78	724.11		175.30%	
Brain extracted	.290 gr	5.46	549.77	343.22	.465	.167	.201	2,121.94	738.11	0.197	176.57%	172.61%
Vial number	23	5.52	546.66	343.43	.475	.163	.206	2,081.60	723.01		176.31%	
		5.45	546.54	342.47	.463	.168	.200	2,111.35	739.68		177.84%	
		5.47	653.12	342.74	.467	.167	.202	2,626.53	733.92		141.84%	
		5.55	549.61	343.55	.479	.161	.208	2,087.21	717.22		174.43%	

Table 3. Continued.

Group 2 - cont.		AES	CPM ^3H	CPM ^{14}C	Efficiencies $^{14}C/^{14}C$	Efficiencies $^{14}C/^3H$	Efficiencies $^3H/^3H$	DPM ^3H	DPM ^{14}C	Injection ratio $^{14}C/^3H$	BUI	Mean BUI
		5.34	386.54	302.90	.447	.174	.192	1,339.11	677.63		245.85%	
		5.32	388.30	302.57	.443	.175	.191	1,407.17	683.00		246.38%	
Rat weight	.308 gr	5.31	491.70	302.17	.442	.175	.190	1,958.21	683.64		177.22%	
Nembutal injected	.176 ml	5.40	383.73	305.06	.456	.170	.197	1,370.56	668.99		247.78%	
Brain extracted	.250 gr	5.43	700.35	310.74	.461	.169	.199	2,946.88	674.06	0.197	116.11%	208.67%
Vial number	29	5.53	386.81	303.06	.477	.163	.207	1,368.36	635.35		235.69%	
		5.48	496.37	314.56	.468	.166	.203	1,895.57	672.14		180.00%	
		5.48	494.44	317.08	.468	.166	.203	1,881.63	677.52		182.78%	
		5.48	384.19	307.65	.468	.166	.203	1,355.02	657.37		246.26%	
		5.07	16,020.00	5,855.97	.400	.191	.173	76,437.98	14,639.93		97.22%	
		5.09	16,158.33	5,855.97	.402	.190	.174	78,157.76	13,467.79		87.47%	
Rat weight	.328 gr	5.10	16,825.00	5,858.69	.404	.189	.175	80,481.03	14,501.71		91.47%	
Nembutal injected	.187 ml	5.11	16,033.33	5,965.21	.407	.188	.176	75,442.61	14,656.54		98.62%	
Brain extracted	.270 gr	5.17	17,342.27	5,186.88	.418	.185	.180	83,593.00	12,408.80	0.197	75.35%	89.78%
Vial number	30	5.01	16,146.66	5,641.48	.390	.195	.170	78,387.76	14,465.33		93.67%	
		5.14	16,046.66	5,285.00	.413	.186	.178	76,778.03	12,796.61		84.60%	
		5.17	16,466.10	5,749.45	.418	.185	.180	77,341.61	13,754.67		90.28%	
		5.13	16,268.33	5,607.82	.411	.187	.177	77,496.27	13,644.33		89.37%	

This table contains the general presurgical and postsurgical information of the sample, the resulting scintillation counts with an Automatic External Standard (AES), the derived Quench Correction Efficiencies and the calculated disintegrations per minute (DPM). The Brain Uptake Index (BUI) is determined by dividing the ratio of $^{14}C/^3H$ DPM found in the brain tissue by the $^{14}C/^3H$ DPM ratio in the injection mixture. The mean BUI is the sum of all BUI's divided by the nine separate counts.

Table 4. Individual sample information and data.

Group 3 - 4 milliwatts		AES	CPM ^{3}H	CPM ^{14}C	Efficiencies $^{14}C/^{14}C$	Efficiencies $^{14}C/^{3}H$	Efficiencies $^{3}H/^{3}H$	DPM ^{3}H	DPM ^{14}C	Injection ratio $^{14}C/^{3}H$	BUI	Mean BUI
		5.19	567.63	18.37	.422	.183	.182	3,075.05	43.53		7.19%	
		5.20	568.70	18.74	.423	.182	.183	3,063.61	44.30		7.34%	
Rat weight	.360 gr	5.20	788.71	18.38	.423	.182	.183	4,266.67	43.45		5.17%	
Nembutal injected	.206 ml	5.18	782.61	17.99	.420	.184	.181	4,280.28	42.83		5.08%	
Brain extracted	.260 gr	5.17	672.88	18.28	.418	.185	.180	3,693.28	43.73	0.197	6.01%	6.09%
Vial number	18	5.19	671.98	18.43	.422	.183	.182	3,648.30	43.67		6.08%	
		5.16	459.82	18.35	.416	.185	.180	2,509.22	44.11		8.92%	
		5.23	893.44	18.00	.429	.180	.184	4,814.62	41.96		4.42%	
		5.19	895.77	18.56	.422	.183	.182	4,877.58	43.98		4.57%	
		5.15	3,839.63	1,987.25	.415	.186	.179	16,474.64	4,788.55		147.54%	
		5.22	3,733.09	1,983.25	.427	.181	.184	15,719.78	4,644.50		149.98%	
Rat weight	.339 gr	5.20	3,852.06	1,982.82	.423	.182	.183	16,387.60	4,678.52		145.20%	
Nembutal injected	.194 ml	5.20	3,748.96	1,762.29	.423	.182	.183	16,342.73	4,166.17		129.40%	
Brain extracted	.220 gr	5.24	3,087.50	1,873.02	.430	.180	.185	12,451.08	4,355.86	0.197	177.58%	143.00%
Vial number	22	5.24	3,633.90	1,090.96	.430	.180	.185	17,174.16	2,537.12		74.99%	
		5.15	3,424.25	1,763.57	.415	.186	.179	14,714.13	4,249.57		146.60%	
		5.23	3,657.46	1,877.26	.429	.180	.184	13,738.59	4,375.90		161.68%	
		5.22	3,657.46	1,983.33	.427	.181	.184	15,308.42	4,644.80		154.02%	
		5.38	1,700.16	539.39	.453	.172	.195	7,668.51	1,190.71		78.82%	
		5.47	1,702.21	646.48	.467	.167	.202	7,282.33	1,384.33		96.49%	
Rat weight	.310 gr	5.45	1,491.04	643.05	.463	.168	.200	6,288.55	1,388.88		112.11%	
Nembutal injected	.177 ml	5.39	1,498.81	537.01	.455	.171	.196	6,617.30	1,180.24		90.54%	
Brain extracted	.260 gr	5.37	1,711.28	644.23	.452	.172	.194	7,557.37	1,425.29	0.197	95.73%	103.36%
Vial number	28	5.52	1,388.87	646.68	.475	.163	.206	5,664.85	1,361.43		121.99%	
		5.47	1,386.50	648.25	.467	.167	.202	5,716.24	1,388.12		123.27%	
		5.38	1,710.94	646.98	.453	.172	.195	7,514.31	1,428.21		96.48%	
		5.46	1,705.96	751.52	.465	.167	.201	7,144.58	1,616.17		114.83%	

This table contains the general presurgical and postsurgical information of the sample, the resulting scintillation counts with an Automatic External Standard (AES), the derived Quench Correction Efficiencies and the calculated disintegrations per minute (DPM). The Brain Uptake Index (BUI) is determined by dividing the ratio of $^{14}C/^{3}H$ DPM found in the brain tissue by the $^{14}C/^{3}H$ DPM ratio in the injection mixture. The mean BUI is the sum of all BUI's divided by the nine separate counts.

Table 5. Individual sample information and data.

Group 4 - 8 milliwatts		AES	CPM ^3H	CPM ^{14}C	Efficiencies $^{14}C/^{14}C$	Efficiencies $^{14}C/^3H$	Efficiencies $^3H/^3H$	DPM ^3H	DPM ^{14}C	Injection ratio $^{14}C/^3H$	BUI	Mean BUI
		5.22	27,567.56	6,902.75	.427	.181	.184	133,921.57	16,165.69		61.27%	
		5.30	27,925.00	6,351.03	.440	.176	.189	134,310.00	14,434.16		54.55%	
Rat weight	.340 gr	5.24	27,289.18	6,578.47	.430	.180	.185	132,623.78	15,298.77		58.56%	
Nembutal injected	.194 ml	5.27	27,627.02	6,904.13	.435	.178	.188	131,924.89	15,871.56		61.07%	
Brain extracted	.290 gr	5.29	27,916.66	6,469.44	.438	.176	.189	133,952.75	14,770.41	0.197	55.97%	61.89%
Vial number	9	5.26	27,737.83	7,433.81	.434	.179	.187	131,934.81	17,128.59		65.90%	
		4.98	27,948.64	7,660.71	.385	.197	.168	143,028.21	19,897.95		70.62%	
		5.29	27,229.72	7,100.70	.438	.176	.189	128,976.03	16,211.64		63.80%	
		5.32	27,945.94	7,458.45	.443	.175	.191	130,887.95	16,836.23		65.29%	
		5.32	5,229.23	150.75	.443	.175	.191	27,066.39	340.29		6.38%	
		5.26	5,659.16	150.75	.434	.179	.187	29,930.37	347.35		5.89%	
Rat weight	.345 gr	5.33	5,325.00	49.48	.445	.174	.191	27,778.69	110.74		2.02%	
Nembutal injected	.197 ml	5.33	5,430.20	43.46	.445	.174	.191	28,341.41	97.66		1.75%	
Brain extracted	.280 gr	5.29	5,658.63	150.41	.438	.176	.189	29,620.05	343.40	0.197	5.88%	4.20%
Vial number	21	5.26	5,439.26	41.25	.434	.179	.187	28,995.99	95.05		1.66%	
		5.31	5,538.29	39.14	.442	.175	.190	29,067.32	88.55		1.55%	
		5.33	5,765.78	43.38	.445	.174	.191	30,098.53	97.48		1.64%	
		5.33	5,312.26	263.15	.445	.174	.191	27,273.66	591.35		11.01%	
		5.57	349.54	121.44	.482	.160	.209	1,479.57	251.95		86.44%	
		5.67	454.69	121.14	.498	.154	.217	1,922.72	243.25		64.22%	
Rat weight	.311 gr	5.60	459.09	121.38	.488	.158	.211	1,989.53	248.73		63.46%	
Nembutal injected	.178 ml	5.69	455.73	120.95	.502	.153	.220	1,903.95	240.94		64.24%	
Brain extracted	.230 gr	5.57	455.75	121.53	.482	.160	.209	1,987.61	252.14	0.197	64.39%	65.12%
Vial number	26	5.61	456.07	121.00	.490	.158	.211	1,976.54	246.94		63.42%	
		5.28	454.28	120.84	.436	.177	.188	2,155.43	277.16		65.27%	
		5.64	562.69	121.22	.494	.156	.213	2,462.02	245.38		50.59%	
		5.55	458.82	121.43	.479	.161	.208	2,009.66	253.51		64.03%	

This table contains the general presurgical and postsurgical information of the sample, the resulting scintillation counts with an Automatic External Standard (AES), the derived Quench Correction Efficiencies and the calculated disintegrations per minute (DPM). The Brain Uptake Index (BUI) is determined by dividing the ratio of $^{14}C/^3H$ DPM found in the brain tissue by the $^{14}C/^3H$ DPM ratio in the injection mixture. The mean BUI is the sum of all BUI's divided by the nine separate counts.

Table 6. Individual sample information and data.

Group 5 - 12 milliwatts		AES	CPM ^3H	CPM ^{14}C	Efficiencies $^{14}C/^{14}C$	Efficiencies $^{14}C/^3H$	Efficiencies $^3H/^3H$	DPM ^3H	DPM ^{14}C	Injection ratio $^{14}C/^3H$	BUI	Mean BUI
		5.35	19,911.76	3,382.72	.449	.173	.193	96,416.58	7,533.90		39.66%	
		5.40	20,347.05	3,603.95	.456	.170	.197	96,464.31	7,903.40		41.59%	
Rat weight	.315 gr	5.35	19,638.46	3,723.18	.449	.173	.193	94,320.83	8,292.16		44.63%	
Nembutal injected	.180 ml	5.24	19,392.30	3,413.26	.430	.180	.185	97,099.95	7,937,81		41.50%	
Brain extracted	.220 gr	5.41	20,125.49	3,725.00	.457	.170	.197	95,125.99	8,150.98	0.197	43.50%	41.10%
Vial number	1	5.48	19,152.94	3,610.07	.468	.166	.203	88,041.63	7,713.82		44.47%	
		5.35	19,690.19	3,163.50	.449	.173	.193	95,706.17	7,045.66		37.37%	
		5.42	19,274.50	3,054.01	.459	.169	.198	91,666.87	6,653.62		36.85%	
		5.45	19,043.13	3,286.37	.463	.168	.200	89,253.35	7,097.99		40.37%	
		5.38	51,715.00	19,574.50	.453	.172	.195	227,090.97	43,210.82		96.59%	
		5.41	52,810.52	19,634.61	.457	.170	.197	230,998.07	42,964.14		94.41%	
Rat weight	.363 gr	5.41	51,565.00	20,003.90	.457	.170	.197	223,978.27	43,772.25		99.20%	
Nembutal injected	.207 ml	5.25	51,905.00	18,909.43	.432	.179	.186	236,934.62	43,771.83		93.78%	
Brain extracted	.200 gr	5.36	50,995.00	20,228.00	.450	.173	.194	222,775.56	44,951.11	0.197	102.43%	96.65%
Vial number	2	5.40	51,240.00	19,694.11	.456	.170	.197	222,831.97	43,188.84		98.38%	
		5.44	51,135.00	19,464.70	.462	.168	.200	220,284.65	42,131.39		97.09%	
		5.38	51,405.00	19,173.07	.453	.172	.195	226,282.87	42,324.66		94.95%	
		5.40	52,000.00	19,039.21	.456	.170	.197	227,929.18	41,752.65		92.99%	
		5.20	596.17	877.32	.423	.182	.183	1,195.03	2,074.04		880.99%	
		5.27	595.54	876.95	.435	.178	.118	1,259.04	2,015.98		812.79%	
Rat weight	.343 gr	5.18	809.13	878.34	.420	.184	.181	2,344.36	2,091.29		452.82%	
Nembutal injected	.196 ml	5.21	597.76	872.18	.425	.181	.183	1,236.67	2,052.19		842.36%	
Brain extracted	.280 gr	5.20	593.80	876.24	.423	.182	.183	1,184.64	2,071.49	0.197	887.63%	731.39%
Vial number	20	5.17	805.80	982.78	.418	.185	.180	2,060.22	2,351.15		579.30%	
		5.25	595.23	874.57	.432	.179	.186	1,251.88	2,024.47		820.89%	
		5.31	813.65	985.88	.442	.175	.190	2,227.95	2,230.50		508.20%	
		5.28	596.10	876.10	.436	.177	.188	1,278.94	2,009.40		797.54%	

Table 6. Continued.

Group 5 - cont.		AES	CPM ^{3}H	CPM ^{14}C	Efficiencies $^{14}C/^{14}C$	Efficiencies $^{14}C/^{3}H$	Efficiencies $^{3}H/^{3}H$	DPM ^{3}H	DPM ^{14}C	Injection ratio $^{14}C/^{3}H$	BUI	Mean BUI
		5.26	9,674.77	2,738.98	.434	.179	.187	45,695.72	6,311.01		70.11%	
		5.23	9,886.23	2,510.27	.429	.180	.184	48,005.27	5,851.45		61.87%	
Rat weight	.356 gr	5.30	9,888.07	2,841.87	.440	.176	.189	46,303.28	6,458.80		70.81%	
Nembutal injected	.203 ml	5.31	8,029.46	2,299.50	.442	.175	.190	37,468.53	5,202.49		70.48%	
Brain extracted	.260 gr	5.26	9,008.03	2,187.56	.434	.179	.187	43,346.47	5,040.46	0.197	59.03%	72.28%
Vial number	25	5.32	8,915.04	2,846.31	.443	.175	.191	40,788.74	6,425.08		79.96%	
		5.27	9,239.63	2,953.06	.435	.178	.188	42,719.41	6,788.64		80.67%	
		5.26	9,673.87	2,953.82	.434	.179	.187	45,217.06	6,806.04		76.41%	
		5.23	8,130.08	2,620.90	.429	.180	.184	38,208.70	6,109.32		81.16%	

This table contains the general presurgical and postsurgical information of the sample, the resulting scintillation counts with an Automatic External Standard (AES), the derived Quench Correction Efficiencies and the calculated disintegrations per minute (DPM). The Brain Uptake Index (BUI) is determined by dividing the ratio of $^{14}C/^{3}H$ DPM found in the brain tissue by the $^{14}C/^{3}H$ DPM ratio in the injection mixture. The mean BUI is the sum of all BUI's divided by the nine separate counts.

Table 7. Individual sample information and data.

Group 6 - 16 milliwatts		AES	CPM ^{3}H	CPM ^{14}C	Efficiencies $^{14}C/^{14}C$	Efficiencies $^{14}C/^{3}H$	Efficiencies $^{3}H/^{3}H$	DPM ^{3}H	DPM ^{14}C	Injection ratio $^{14}C/^{3}H$	BUI	Mean BUI
		4.89	673.32	124.34	.367	.204	.161	3,752.80	338.80		45.83%	
		4.79	670.41	124.34	.349	.211	.154	3,865.19	356.28		46.79%	
Rat weight	.287 gr	4.88	784.89	124.46	.366	.204	.161	4,444.22	340.05		38.84%	
Nembutal injected	.164 ml	4.80	890.71	124.37	.350	.210	.155	5,265.10	355.34		34.26%	
Brain extracted	.270 gr	4.81	785.19	124.40	.352	.209	.156	4,559.81	353.41	0.197	39.34%	39.07%
Vial number	5	4.79	783.39	124.15	.349	.211	.154	4,599.55	355.73		39.26%	
		4.80	784.57	124.72	.350	.210	.155	4,578.97	356.34		39.50%	
		4.83	892.61	124.03	.356	.208	.157	5,223.82	348.40		33.86%	
		4.80	891.64	123.53	.350	.210	.155	5,274.32	352.94		33.97%	
		5.13	1,273.41	372.20	.411	.187	.177	6,237.63	905.60		73.70%	
		5.20	1,053.79	375.87	.423	.182	.183	4,874.70	888.58		92.53%	
Rat weight	.326 gr	5.24	1,947.03	378.89	.430	.180	.185	9,667.14	881.14		46.27%	
Nembutal injected	.186 ml	5.18	1,051.23	265.34	.420	.184	.181	5,165.69	631.76		62.08%	
Brain extracted	.240 gr	5.19	1,944.66	267.04	.422	.183	.182	10,048.68	632.80	0.197	31.97%	67.99%
Vial number	11	5.22	1,275.20	375.58	.427	.181	.184	6,065.22	879.58		73.61%	
		5.15	1,163.89	482.70	.415	.186	.179	5,293.58	1,163.13		111.54%	
		5.17	1,727.47	379.81	.418	.185	.180	8,663.17	908.64		53.24%	
		5.23	1,381.64	376.47	.429	.180	.184	6,651.52	877.55		66.97%	
		5.39	58,661.11	11,360.00	.455	.171	.196	277,508.92	24,967.03		45.67%	
		5.34	57,744.44	11,035.29	.447	.174	.192	292,070.05	24,687.45		42.91%	
Rat weight	.358 gr	5.40	57,683.33	12,128.57	.456	.170	.197	269,856.39	26,597.74		50.03%	
Nembutal injected	.205 ml	5.36	58,761.11	11,592.94	.450	.173	.194	300,537.47	25,762.09		43.51%	
Brain extracted	.280 gr	5.42	58,855.55	11,375.29	.459	.169	.198	276,097.27	24,782.77	0.197	45.56%	42.14%
Vial number		5.42	57,638.88	11,922.35	.459	.169	.198	268,935.20	25,974.62		49.03%	
		5.44	59,888.25	11,026.19	.462	.168	.200	279,393.65	23,866.21		43.36%	
		5.43	58,438.88	12,560.24	.461	.169	.199	288,745.32	5,790.27		10.18%	
		5.47	58,650.00	12,125.00	.467	.167	.202	268,881.58	25,963.60		49.02%	

This table contains the general presurgical and postsurgical information of the sample, the resulting scintillation counts with an Automatic External Standard (AES), the derived Quench Correction Efficiencies and the calculated disintegrations per minute (DPM). The Brain Uptake Index (BUI) is determined by dividing the ratio of $^{14}C/^{3}H$ DPM found in the brain tissue by the $^{14}C/^{3}H$ DPM ratio in the injection mixture. The mean BUI is the sum of all BUI's divided by the nine separate counts.

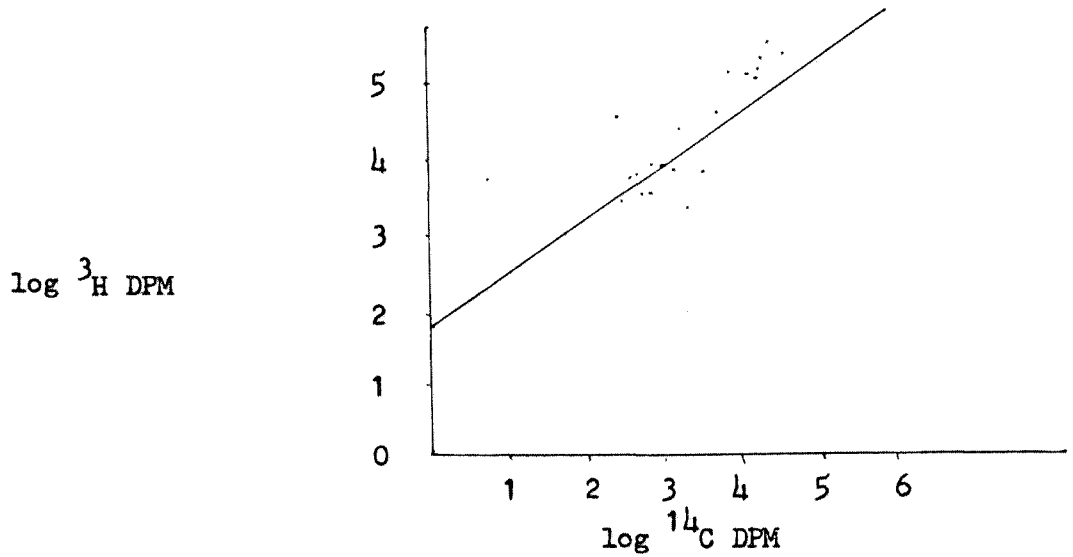

Figure 4. Linear regression of the logarithms of the ^{3}H DPM found in the brain tissue to the logarithms of the ^{14}C DPM found in the brain tissue. The line has a slope of 0.75, the ordinate intercept is 1.72, and the regression coefficient is 0.81.

Table 8. Logs of ^{3}H DPM and ^{14}C DPM.

Vial number	DPM's ^{3}H	DPM's ^{14}C	Group number	Logs ^{3}H	Logs ^{14}C
18	4,345.13	43.51	3	3.638	1.639
21	28,685.82	243.65	4	4.485	2.387
26	1,987.45	251.11	4	3.298	2.400
14	4,087.77	299.40	2	3.611	2.476
5	4,618.20	350.81	6	3.664	2.545
29	1,731.39	669.97	2	3.238	2.826
23	2,156.92	729.84	2	3.334	2.863
11	6,963.04	863.20	6	3.843	2.936
28	6,828.23	1,373.71	3	3.834	3.138
10	5,374.35	1,485.54	2	3.730	3.172
6	23,907.00	1,767.11	1	4.379	3.247
20	1,559.86	2,102.28	5	3.193	3.323
22	15,367.90	4,272.22	3	4.187	3.631
25	43,083.69	6,110.37	5	4.634	3.786
1	93,788.41	7,592.15	5	4.972	3.880
30	78,235.12	13,815.08	2	4.893	4.140
31	69,617.54	15,780.76	1	4.843	4.198
9	113,506.65	16,290.56	4	5.055	4.212
3	178,868.16	19,956.49	1	5.253	4.300
12	280,225.06	23,154.64	6	5.448	4.365
2	226,567.31	43,118.63	5	5.355	4.635

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